DOI: https://doi.org/10.5114/ait.2021.103513

A sudden presentation of abdominal compartment syndrome

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Dear Editor,

Abdominal compartment syndrome (ACS) is defined as sustained intra-abdominal pressure (IAP) exceeding 20 mm Hg, which causes end-organ damage due to impaired tissue perfusion, as with other compartment syndromes [1, 2]. This dysfunction can extend beyond the abdomen to other organs like the heart and lungs. ACS is most commonly caused by trauma or surgery to the abdomen. It is characterised by interstitial oedema, which can be exacerbated by large fluid shifts during massive transfusion of blood products and other fluid resuscitation [3]. Normally, IAP is nearly equal to or slightly above ambient pressure. Intra-abdominal hypertension is typically defined as abdominal pressure greater than or equal to 12 mm Hg [4]. Initially, the abdomen is able to distend to accommodate the increase in pressure caused by oedema; however, IAP becomes highly sensitive to any additional volume once maximum distension is reached. This is a function of abdominal compliance, which plays a key role in the development and progression of intra-abdominal hypertension [5]. Surgical decompression is required in severe cases of organ dysfunction – usually when IAPs are refractory to other treatment options [6]. Excessive abdominal pressure leads to systemic pathophysiological consequences that may warrant admission to a critical care unit. These include hypoventilation secondary to restriction of the deflection of the diaphragm, which results in reduced chest wall compliance. This is accompanied by hypoxaemia, which is exacerbated by a decrease in venous return. Combined, these consequences lead to decreased cardiac output, a V/Q mismatch, and compromised perfusion to intra-abdominal organs, most notably the kidneys [7]. Kidney damage can be prerenal due to renal vein or artery compression, or intrarenal due to glomerular compression [8] – both share decreased urine output as a manifestation. Elevated bladder pressure is also seen from compression due to increased abdominal pressure, and its measurement, via a Foley catheter, is a diagnostic hallmark. Sustained intra-bladder pressures beyond 20 mm Hg with organ dysfunction are indicative of ACS requiring intervention [2, 8]. ACS is an important aetiology to consider in the differential diagnosis for signs of organ dysfunction – especially in the perioperative setting – as highlighted in the case below.

In this case, an 8-year-old girl had ACS that was only detected by an inability to ventilate at the end of a surgery. The ACS was initially misdiagnosed due to its sudden presentation which suggested a more acute aetiology such as pneumothorax.

This study was conducted in accordance with all relevant institutional and ethical review board policies and was approved by the Rutgers New Jersey Medical School Institutional Review Board. Written informed consent was obtained from the patient's parent.

Anaesthesiol Intensive Ther 2021; 53, 1: 1–4

Received: 16.09.2020, **accepted:** 24.01.2021

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An 8-year-old girl with weight 25 kg and height 132 cm was an unrestrained passenger in an automobile collision and sustained massive trauma with fractures to her left scapula, bilateral femurs, the L1–L5 vertebrae, and her right tibia-fibula. Upon presentation to the hospital, she had bilateral pneumothoraces and required emergent exploratory laparotomy to establish haemostasis and wash-out of hemoperitoneum from numerous abdominal soft-tissue injuries including hepatic and splenic lacerations as well as bleeding from the left ovary. This was followed by primary closure of the abdomen during the same first operation. The patient remained intubated post-operatively pending further surgical intervention to achieve internal fixation of the femurs and the tibia-fibula. This second surgery was scheduled for the following day and is the focus of the present report.

A physical examination of the abdomen prior to this second operation was unremarkable with a soft abdomen (as felt by the anaesthesiologist and corroborated by the surgical documentation). Prior to the start of the second surgery, in the operating room, peak airway pressures were elevated at ~45 mm Hg with an upsloping $EtCO_2$ capnography; this was diagnosed as bronchospasm and treated with an albuterol nebuliser with a subsequent decrease in peak airway pressures to the 20s (mm Hg). Orthopaedic intervention on the right leg then proceeded and was complete by the third hour of surgery, at which point the case proceeded to the left leg; each leg was elevated while being worked on. The patient had mild hypotension and 200 mL of blood loss, which was addressed by administering an additional unit of packed red blood cells. The case was otherwise uneventful with steady peak airway pressures in the mid-20s and $EtCO_2$ in the mid-30s (mm Hg). Upon conclusion of the case, in the fourth hour of surgery, while dressings were being applied, the $EtCO_2$ rose to the 50s (mm Hg), concomitant with an increase in peak airway pressure from the 20s to the 40s (mm Hg). This occurred relatively suddenly, and peak pressures rapidly increased beyond 50 mm Hg and progressed to a complete loss of ventilation with no $EtCO_2$.

The patient maintained oxygenation with 100% SpO_2, but no ventilation was achieved, no breath sounds could be auscultated, and no chest movement was observed. At this time, the patient was already paralysed because maintaining the intubated airway post-operatively was planned. After confirming tube position by laryngoscopy, reinspection of the endotracheal tube cuff, and suctioning via the endotracheal tube, we suspected recurrence of bronchospasm and treated it accordingly with 200 µg of epinephrine twice to no avail. Help was called upon from anaesthesia colleagues, and the trauma surgical team came to the OR. At that time intermittent breath sounds were briefly auscultated on the left side. Needle decompression for a tension pneumothorax was considered but ruled against given the presence of intermittent return of $EtCO_2$ with peak values of 52 mm Hg. A decision was made to perform a quick chest X-ray given that the machine and radiology technician were already in the operating room in anticipation of a routine post-orthopaedic procedure imaging. Radiology revealed no obvious signs of a tension pneumothorax at which point, as expected, the patient eventually began to desaturate after maintaining oxygenation with little to no ventilation for about 10 minutes. Tension pneumothorax was still at the top of the differential diagnosis at the time, and a right needle decompression was then performed by the anaesthesiologist to rule-out tension pneumothorax more definitively – minimal air and blood released from the needle. Transcutaneous pacing pads were applied to the patient in anticipation of eventual cardiac arrest from worsening respiratory acidosis. Bicarbonate was administered as a temporising measure to counter the acidosis. ABG later showed a $PaCO_2$ of 150 mm Hg. The paralysis was deepened in order to preserve oxygen by minimising peripheral oxygen consumption. Eventually a physical examination noted the abdomen to be stiff and ACS rose to the top of the differential. Emergent decompressive laparotomy resulted in rapid return of ventilation with normalisation of peak airway pressures and tidal volumes. Visual inspection showed a grossly oedematous bowel and confirmed ACS as the aetiology for the acute respiratory decompensation. The abdomen could not be closed without recurring compensation and accordingly was left open with a protective occlusive barrier and the patient was transported to the paediatric ICU. Time progression of $EtCO_2$ and peak airway pressures, along with blood gas and electrolyte data, are given in Figure 1 and Table 1, respectively. The abdomen was closed 8 days after the decompressive laparotomy, and the patient was discharged to home 11 days after closure, having spent 20 days in hospital.

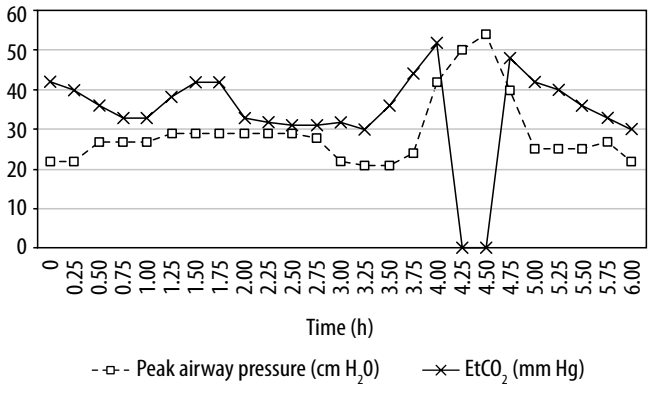

FIGURE 1. Evolution of $EtCO_2$ and peak airway pressure over time

This case is unique in its sudden presentation, which contrasts with other reported cases of ACS in children [9–11]. A sudden manifestation of ACS is likely to be preceded by a gradual increase in IAP over a long period of time; the present case gives an archetypical example of this. Acute and sub-acute ACS usually progress for several hours to days before necessitating surgical decompression [12]. The incidence of ACS appears to correlate with intraoperative resuscitation volume in children [13]. Hyperacute ACS has been described in adults after massive fluid resuscitation during extra-abdominal surgery [14]. In adults, mortality from ACS substantially decreased over the years while the mortality in children remained the same – this is possibly attributed to failure to promptly recognise the condition in children [15]. The paediatric physiology sometimes has the remarkable ability to compensate and mask a pathology until it simply cannot; extra vigilance is warranted in this population.

Accordingly, we recommend evaluation of the abdomen by physical exam during the pre-operative anaesthesia assessment and consideration for periodic measurement of IAP for all patients with recent history of abdominal trauma. There should be a low threshold for surgical consultation when compliance of the abdomen is in question, e.g. non-reassuring exam or concerning history. This case emphasises the importance of physical examination skills in the evaluation of the abdomen preoperatively and intraoperatively. Percussion and palpation of the abdomen can reveal a dense abdomen. It is likely that this patient had elevated IAP prior to the surgery and that this was undiagnosed and subsequently worsened by intraoperative fluid administration, increased bowel oedema, and higher IAP. Consideration for ACS should be made in the context of recent abdominal surgery, in orthopaedic cases with recent deceleration injuries, and in burn victims [16].

Intraoperatively, in the setting of vasodilation from anaesthetics that often lead to a relative hypovolaemia, the diagnosis of ACS can be harder to make. Decreased urine output from kidney injury secondary to abdominal compartment syndrome can easily be mistaken as a sign of hypovolaemia and wrongly treated with more fluid rather than fluid restriction. Cardiac output decreases in patients with ACS due to a decrease in preload. However, unlike other scenarios in which a decreased preload is typically treated with fluid administration, fluid in ACS is likely to worsen the situation by exacerbating the underlying pathophysiology.

TABLE 1. Blood gas and electrolyte panel

Blood gas/Electrolyte	Pre-Op	Intra-op 1	Intra-op 2	Post-op
pH	7.377	6.608	7.211	7.288
PCO_2, mm Hg	41	137	61	45
PO_2, mm Hg	80	95	145	84
Temperature, °F	99.3	98.6	98.6	97.4
BE, mmol L^{-1}	-1.5	-15	-4.3	-5.4
HCO_3, mmol L^{-1}	24	21	24	21
CO_2 CT, mmol L^{-1}	25	25	26	23
O_2 Sat, %	94.7	91	97.8	95.5
pH temp	7.37	6.808	7.211	7.298
PCO_2 temp, mm Hg	42	137	61	44
PO_2 temp, mm Hg	82	95	145	80
$\%FiO_2$, %	30	100	100	40
WB sodium, mmol L^{-1}	135	136	138	135
WB potassium, mmol L^{-1}	3.7	4.6	3.8	4.3
WB chloride, mmol L^{-1}	111	110	109	107
WB glucose, mmol L^{-1}	118	155	153	112
WB lactic acid, mmol L^{-1}	1.4	1.8	2.3	1.4
Ionized calcium, mmol L^{-1}	1.12	1.17	1.06	1.19

The intermittent ventilation that we were experiencing during the patient's collapse may be explained by the mechanics of the anaesthesia machine circuit. The adjustable pressure-limiting (APL) valve is designed to prevent airway pressures above its set value during manual ventilation mode. However, the APL is not a hard stop – manual squeezing of the ventilation bag, particularly when aggressive, can easily lead to brief airway pressures above the APL setting. In this case we were using a Dräger machine with an APL valve with a maximum setting of 70 mm Hg. We believe that we were ventilating the patient briefly only when the airway pressures were exceeding the 70-mm Hg threshold.

IAP may aid in diagnosing ACS perioperatively. This is likely to be most feasible through a Foley catheter [17]. Intragastric catheters have been used but carry potential hazards and tend to produce inaccurately low measurements [18]. Abdominal perfusion pressure (APP) is the ultimate concern, and it has been shown to be a more accurate predictor of visceral perfusion than IAP alone [12]. Bearing in mind that APP equals MAP – IAP, maintaining MAP is a vital part of the treatment. An APP ≥ 60 mm Hg has been suggested to increase survival in patients with ACS and intra-abdominal hypertension, while a causal relationship has not been established; hence, further study may prove informative [12, 19]. It is possible that the patient's abdominal compliance was initially improved with administration of the neuromuscular blockade and that this may have contributed to masking the problem in its early stages [20, 21].

The use of colloids may reduce the incidence of ACS by reducing volume

requirements for resuscitation [22]. It is important to remember that blood products are fluids. Perhaps periodic IAP measurements in the OR should be considered for patients with abdominal trauma and concern for ACS. We posit that this patient developed ACS from her trauma and her initial surgery, and that administration of additional crystalloid and blood during her second surgery exacerbated her condition.

This case teaches us that ACS can present as a sudden respiratory decompensation in the paediatric population and should be considered early in the differential when diagnosing acute respiratory failure in surgical patients at high risk of developing ACS.

ACKNOWLEDGEMENTS

1. Assistance with the article: none.
2. Financial support and sponsorship: none.
3. Conflicts of interest: none.

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